Left ventricular reconstruction: Early and late results

Lynda L. Mickleborough, MD
Naeem Merchant, MD
Joan Ivanov, PhD
Vivek Rao, MD
Susan Carson, AHT

Dr Mickleborough

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From the University of Toronto, Toronto, Ontario, Canada.

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Address for reprints: Lynda L. Mickleborough, MD, 1221 Gervais Rd, RR #1, Waubaushene, Ontario L0K 2C0, Canada (E-mail: l.mickleborough@on.aibn.com).

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Objectives: In patients with coronary disease and poor left ventricular function, ventricular reconstruction with revascularization is a surgical option. Details of patient selection and optimal surgical technique are still debated. This study reports results achieved with ventricular reconstruction in 285 patients who had akinesia or dyskinesia associated with relative wall thinning.

Methods: Data were prospectively collected. Reconstruction on the beating heart was accomplished by a modified linear closure plus septoplasty, when indicated, (dyskinetic septum). Preoperatively, 237 (83%) were in symptom class III or IV with congestive heart failure (n =174; 61%), angina (n = 157; 55%), or ventricular tachycardia (n = 107; 38%). Average ejection fraction was 24% ± 11%, and 144 (51%) had preoperative grade 2+ mitral regurgitation. Operative procedures included coronary artery bypass grafting in 262 (92%), septoplasty in 64 (22%), ablation of ventricular tachycardia in 118 (41%), and a mitral valve procedure in 6 (2%).

Results: Operating room mortality was 2.8%. Perioperative support included intraaortic balloon pumping in 49 (17%) and inotropic drugs in 154 (54%). During a mean follow-up of 63 ± 48 months, 8 patients required transplantation (interval of 49 ± 41 months), 2 needed mitral valve replacement, and 9 required use of an implantable cardioverter-defibrillator for ventricular tachycardia. At 1, 5, and 10 years actuarial survivals were 92%, 82%, and 62%. Freedom from sudden death was 99%, 97%, and 94%. Among survivors, symptom class improved in 140 of 208 patients (67%), mean improvement 1.3 ± 1.1 functional class per patient. Average increase in ejection fraction postoperatively was 10% ± 9%.

Conclusions: Using wall thinning as a criterion for patient selection, left ventricular reconstruction can be performed with low operative mortality, provides good control of symptoms, excellent long-term survival, and freedom from sudden death. This approach should be considered in all patients with coronary disease, poor left ventricular function, and relative wall thinning.

Treatment of ischemic heart disease continues to be a challenge with more patients presenting with congestive heart failure. In patients with coronary disease and a previous infarct, the goal of surgery should be not only to prevent ongoing ischemia, but also to minimize the negative effects of the infarct on ventricular structure and function. After infarction, necrotic muscle is replaced by fibrous tissue. When significant scarring occurs, an area of relative thinning often results along the

distribution of the infarct vessel (elliptical in shape). The amount of thinning and compensatory ventricular dilatation that occurs is highly variable.

For many years, left ventricular (LV) aneurysm resection has been recommended in patients with coronary disease as a treatment for heart failure, angina, and thromboembolic complications or to control ventricular arrhythmias. The original procedure as applied to discrete dyskinetic aneurysms included excision of the thin-walled sac, leaving behind a rim of scar to facilitate closure, which was accomplished in a linear fashion. Technical modifications have been advocated including the purse-string technique of Jatene,[1] the endoaneurysmorrhaphy technique originally described by Cooley,[2] and the endoventricular circuloplasty of Dor and coworkers.[3] Recently, these techniques have been applied in patients without a discrete aneurysm (ventricular reconstruction) as a treatment for ischemic cardiomyopathy.[4-6]

In the 1980s we began resecting areas of scar mixed with viable muscle in patients with ventricular arrhythmias. These areas were thinner than normal but were not classic aneurysms as described in the literature. By resecting these portions of the wall we could approach arrhythmogenic sites, which almost always corresponded to areas of septal scarring. The repair involved patch exclusion of any aneurysmal portion of the septum. Encouraged by our results in 1994, we advocated a liberal approach to ventricular reconstruction.[7] Goals are to resect or exclude thinned, nonfunctioning parts of the ventricle and to restore size and shape toward normal as much as possible. We described this new technique, which combines patch septoplasty, when indicated, with a modified linear closure and recommended it for use in patients with akinetic or dyskinetic scar.[8] This article updates our experience and describes results achieved. Whether the procedure is called aneurysm resection, heart reduction surgery, ventricular surgical remodeling, or restoration, the principles are the same. We have used preoperative and postoperative multiple gated acquisition (MUGA), echocardiography and more recently magnetic resonance imaging (MRI) to provide objective evidence of improvement in ventricular and mitral valve function.

Methods

Between 1983 and 2002, 285 consecutive patients underwent ventricular reconstruction by a single surgeon (L.M.). In 3 cases with extensive calcification, a free wall patch was needed to achieve closure without distorting the ventricular chamber. These cases have been excluded from this series. In 282 patients tailored wall excision and septoplasty, when indicated, was followed by a modified linear closure. Patients were selected for the operation. Those with right ventricular (RV) hypokinesis or severe pulmonary hypertension (pulmonary artery pressures at the systemic level) and those with diffuse disease in all coronary distributions were considered inoperable. Until recently, associated severe mitral regurgitation (MR) was considered a relative contraindication. On ventriculography, gross chamber dilatation or extensive wall motion abnormalities were not considered a contraindication so long as reasonable contractile function was preserved in some portion of the ventricle. Since 1988, detailed wall motion analysis was possible in 161 patients by a center-line method.[9] The extent of asynergy was calculated as a percentage length of the LV perimeter showing a fractional shortening below 2 SDs from normal mean values.[9] Angiography provides no information with regard to extent or distribution of wall scarring and thinning. In dilated poorly functioning hearts, it was often difficult to accurately assess size and function at the apex by 2D echocardiographic techniques. In the past we have used needle aspiration to evaluate wall thinning intraoperatively.[8] Since 1998 we have used MRI to preoperatively assess ventricular shape, volume, regional wall motion, and thinning. This allowed us to decide whether a patient with poor LV function was a candidate for revascularization alone (no thinning) or in combination with surgical reconstruction. Some evaluation of ventricular size was possible in 234 of 253 anterior scars (92%): echocardiographic end-diastolic dimension was used in 158 (62%), ventriculographic analysis in 179 (71%), or MRI in 41 (16%). Preoperative and postoperative MRI data were used to evaluate changes in volume and shape by the eccentricity index.[10]

Operative Procedure

Our operative procedure, which includes tailored scar excision, septoplasty, when indicated, and modified linear closure, has been described.[8] We use MRI or needle aspiration to determine whether akinesis or dyskinesis in the free wall corresponds to an area of hibernating myocardium that may benefit from revascularization or to an area of scarred, thinned wall that should be resected. Once thinning is established, a small incision is made and any clot present is removed. Only then is a vent inserted. With the unloaded heart open and beating, the surrounding wall is palpated and its ability to contract (regional wall thickening) assessed. All thinned, nonfunctioning portions are considered for resection. The size and shape of the cavity that will be left behind can be evaluated. Excision is planned to remove as much nonfunctioning wall as possible and to restore ventricular size and shape toward normal by the modified linear closure.[8] One can avoid creating too small a chamber by preserving or reestablishing normal spatial relationships between the papillary muscle insertions and the septum.

In patients with significant septal scarring, a visually directed endocardial excision and cryoablation at the periphery is carried out.[11] When part of the septum is aneurysmal (clearly bulging into the RV), a patch septoplasty is performed. This excludes the aneurysmal septum from the remaining LV cavity, as shown in Figure 1. The anterior edge of the patch is incorporated into the modified linear closure as previously described.[8] Length of the closure is plicated in the repair. In this manner the LV “apex” is recreated adjacent to the RV apex and ventricular shape is restored toward normal (Figures 1 and 2). After repair, revascularization is carried out with a graft to the proximal left anterior descending coronary artery whenever possible. We use retrograde cardioplegia to protect the LV and antegrade cardioplegia or infusion down a right graft to protect the RV.

Follow-up

Follow-up was completed in 2002 by telephone contact with the patient or the referring physician. Preoperative and postoperative studies were done by MUGA in 228 (89%) to assess LV ejection fraction (LVEF), by echocardiography in 203 (71%) to assess MR, and by MRI in 29 (10%) to assess ventricular volume and shape.

Statistical Analysis

The SAS (SAS Institute, Inc, Cary, NC) and BMDP (BMDP Software, Los Angeles, Calif) programs were used for statistical analysis. Patient characteristics were compared by means of the *t* test for continuous variables and the χ^2 or Fisher exact test for categorical variables. Results are presented as mean ± SD in the text and tables and mean ± SE in the figures. Actuarial survival was calculated by the Kaplan-Meier statistic. A Wilcoxon test was used to evaluate differences in survival among patient subgroups. A good long-term result, defined as 5-year survival without the need for transplantation or repeated hospitalizations (more than one) for heart failure was evaluated multivariately by a Cox proportional hazards model with the following building strategy. Variables with a univariate *P* value of less than .25 or those of known biologic importance but failing to meet the critical α level were submitted for consideration to the multivariate analysis by stepwise selection.

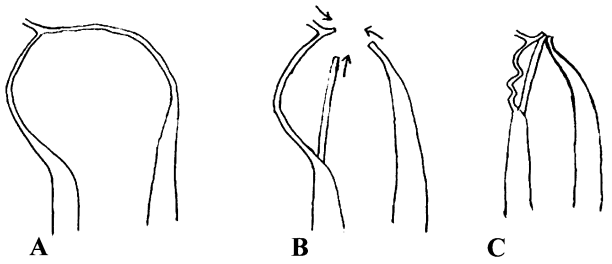

Figure 1. Technique of septal aneurysm patch exclusion. A, Apical aneurysm with significant thinning and aneurysmal involvement of distal septum. B, Pericardial patch sewn to the preserved normal portion of the septum on three sides. The anterior edge of the patch is pulled tight and incorporated into the anterior modified linear closure as indicated by the *arrows*. C, The patch effectively excludes the aneurysmal portion of the septum from the residual LV cavity and helps restore more normal conical shape of the ventricle. (Reprinted from Mickleborough LL, Merchant N, Provost Y, Carson S, Ivanov J. Ventricular reconstruction for ischemic cardiomyopathy. *Ann Thorac Surg*. 2003;75:S6-12; published with permission.)

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Results

Patient Population

Subgroup characteristics—anterior compared with posterior and akinetic compared with dyskinetic—are listed in Appendix 1. There were 285 patients whose ages ranged from 22 to 78 years (mean, 60 ± 10 years). There were 38 female patients (17%). Mean delay from infarct to repair was 54 ± 71 months (median 12 months). Resection was anterior in 253 (89%) and posterior in 32 (11%). Major indications for surgery were often multiple and included class III or IV congestive heart failure, 174 (61%); class III or IV angina, 157 (55%); ventricular tachycardia, 108 (38%); and embolism, 6 (2%). Most patients (n = 237, 83%) were in symptom class III or IV (angina, congestive heart failure, or both). Double or triple vessel disease was present in 239 (84%). The distribution of preoperative EF is shown in Figure 3. Two hundred sixty-eight patients (94%) had an EF less than 40% (mean, 24% ± 11%). Of patients analyzed, 97 (60%) had akinesia and 64 (40%) had dyskinesia. Mean asynergy was 54% ± 15% for dyskinetic scar and 54% ± 18% for akinetic scar. Ventricular volume was grossly enlarged (end-diastolic dimension greater than 63 mm or end-diastolic volume index (EDVI) greater than 100 mL/m^2) in 101 of 253 patients (40%).

At operation, associated procedures included clot removal in 73 (26%), patch septoplasty in 64 (22%), arrhythmia ablation in 118 (41%), mitral valve replacement in 3 (1%), mitral annuloplasty in 3 (1%), and coronary artery bypass grafting in 264 (93%), with an average of 3.0 ± 1.0 grafts per patient. In 19 (7%) the entire procedure was carried out on the beating heart. In 266 the average cross-clamp time was only 63 ± 25 minutes. Average pump time was 171 ± 52 minutes. All patients were successfully weaned from bypass, but 141 (50%) required inotropic support and 49 (17%) required an intra-aortic balloon pump.

There were 8 hospital deaths (mortality 2.8%). Two were related to adult respiratory distress syndrome in patients receiving amiodarone. Six were related to congestive heart failure and ventricular arrhythmias. Five patients (1.8%) had perioperative infarcts. Eight patients (3%) required reoperation for bleeding. There were 3 (1%) sternal infections, 2 (0.7%) transient ischemic attacks, and 1 (0.4%) stroke.

During follow-up, which extends to 19 years (mean, 63 ± 48 months), there were 69 late deaths: congestive heart failure, 39; sudden death, 8; ventricular arrhythmias, 1; stroke, 1; renal failure, 3; myocardial infarction; 4; chronic obstructive pulmonary disease, 1; brain aneurysm, 1; and cancer, 6. Two patients died after a subsequent transplant and 1 patient died after a subsequent mitral valve replacement. The cause of death was unknown in 2 patients.

The actuarial survival curves (including hospital deaths) for the entire population and various subgroups are shown in Figure 4. Overall survival was 92% at 1 year, 82% at 5 years, and 62% at 10 years. Among survivors, 7 patients required cardiac transplantation (mean interval 48 ± 40 months). Two required mitral valve replacement, 18 required a pacemaker for bradyarrhythmias, and 9 received an implantable cardioverter-defibrillator. Excluding patients receiving a defibrillator, freedom from sudden death or recurrent ventricular tachycardia was 99%, 97%, and 94% at 1, 5, and 10 years.

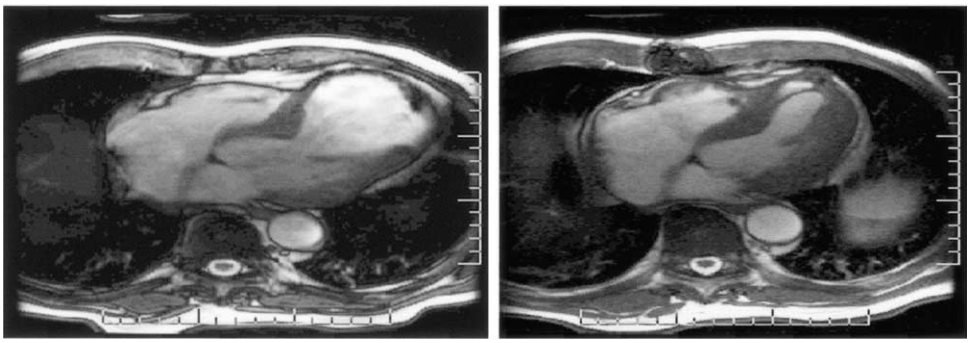

Figure 2. Preoperative *(left)* and postoperative magnetic resonance imaging. Preoperatively, an anteroapical aneurysm involves the distal portion of the septum; the apex is globular in shape. Postoperatively, a septal patch has excluded the aneurysmal septum and is incorporated anteriorly into the modified linear closure. The space between the patch and septal aneurysm has been filled with clot. Ventricular volume is decreased and the shape of the apex has been restored toward normal; namely, it is more conical. (Reprinted from Mickleborough LL, Merchant N, Provost Y, Carson S, Ivanov J. Ventricular reconstruction for ischemic cardiomyopathy. *Ann Thorac Surg*. 2003;75:S6-12; published with permission.)

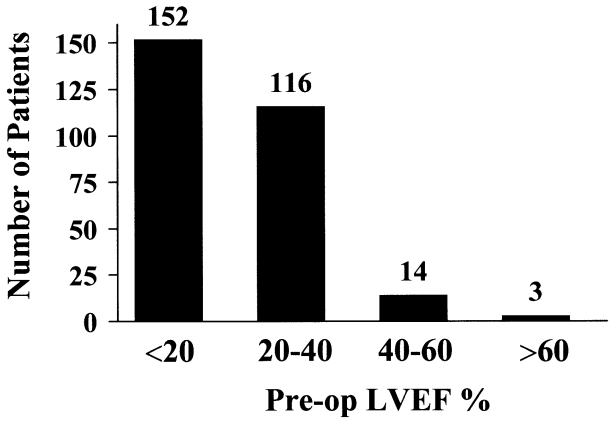

Figure 3. Histogram showing distribution of preoperative LVEF among our patient population.

According to multivariable analysis (Table 1), predictors of poor 5-year result included EF less than 20%, congestive heart failure, preoperative ventricular tachycardia, and a history of hypertension. Poor outcome was not related to age, sex, extent of coronary disease, MR, presence or absence of septoplasty, crossclamp time, or pump time. Because assessment of wall motion and ventricular volume was not available in all patients, we could not enter these variables into the model. However, at 5 years 26 of 78 patients (33%) with a grossly enlarged heart had poor results versus 11 of 59 (19%) with a small heart ($P = .080$). Of patients with dyskinesis, 7 of 39 (18%) had a poor 5-year result versus 18 of 66 (27%) of those with akinesis ($P = .347$).

Among survivors, 140 of 208 (67%) were symptomatically improved, 76 (37%) symptom class I and 60 (29%) symptom class II. Average increase in symptom class was 1.3 ± 1.1 classes per patient ($P < .000$). Preoperative and postoperative MUGA or MRI assessment was available in 178 of the patients with an anterior aneurysm. There was a statistically significant increase in EF (paired *t* test), with a mean change of $10\% \pm 9\%$ ($P < .000$). MRI in 41 patients revealed a mean preoperative EDVI of 133 ± 52 mL/m^2 and a mean end-systolic volume index (ESVI) of 97 ± 49 mL/m^2. Postoperative mean EDVI was 103 ± 35 mL/m^2 and mean ESVI 65 ± 31 mL/m^2, mean change EDVI 29 ± 37 mL/m^2 and ESVI 32 ± 39 mL/m^2 ($P < .000$). The mean diastolic eccentricity index was 0.78 ± 0.06 preoperatively versus 0.74 ± 0.11 postoperatively ($P = .039$). Average systolic eccentricity index was 0.83 ± 0.06 preoperatively versus 0.81 ± 0.07 postoperatively ($P = .118$). Preoperative and postoperative echocardiograms were performed in 203 patients. Of these 129 (64%) had grade 2+ or more MR preoperatively. In 74 of 129 (57%) the MR was improved by at least 1 grade postoperatively.

Discussion

This article updates results achieved in a large series of patients with akinetic or dyskinetic areas of relative wall thinning in whom ventricular reconstruction was accomplished by a modified linear closure technique. Patch septoplasty is an important part of the procedure when a portion of the septum is aneurysmal. A knowledge of normal ventricular size and geometry is essential before using this technique. Wall excision is tailored to preserve or reestablish the normal spatial relationships between papillary muscle insertion sites and the septum and to restore cavity size and shape as much toward normal as possible. This avoids creating too small a cavity with the repair. We believe, as does Jatene,[1] that completing the repair on the beating heart has the advantages of minimizing the ischemic time and

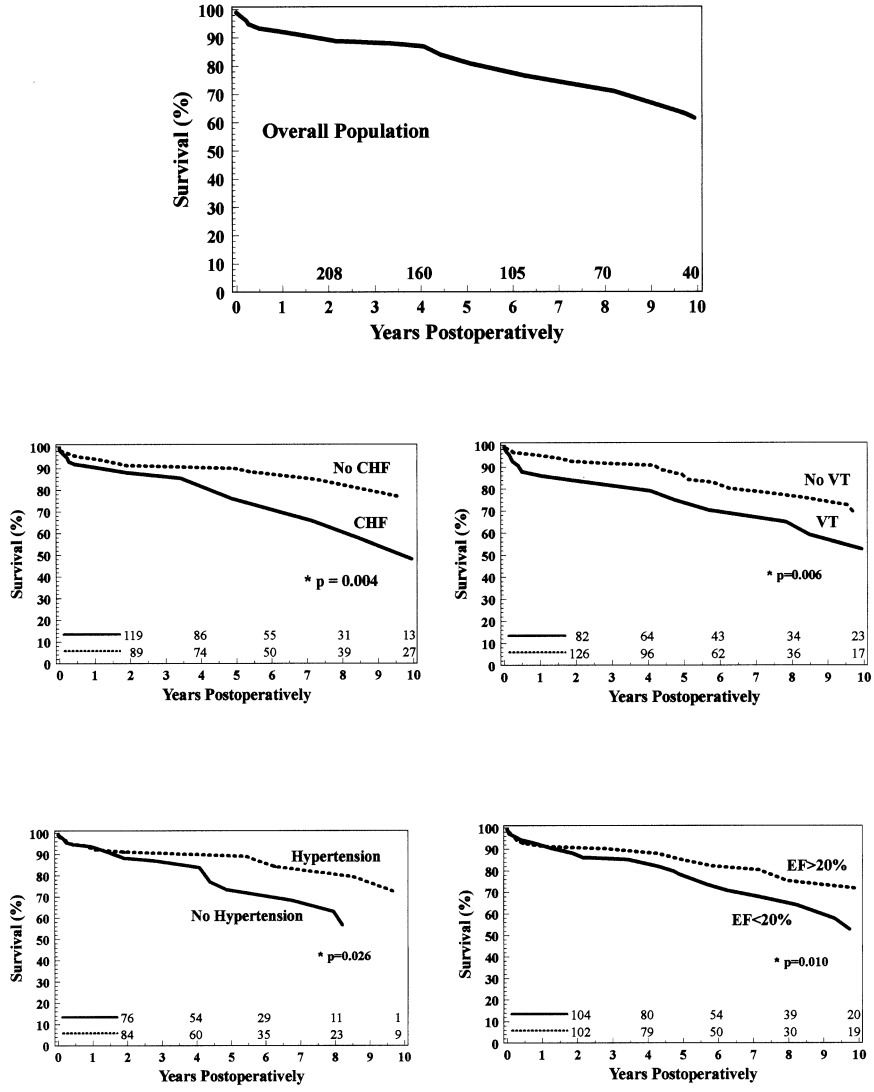

Figure 4. Actuarial survival after ventricular reconstruction for overall population and for subgroups with and without congestive heart failure *(CHF),* ventricular arrhythmias *(VT),* hypertension, or EF less than 20%. Asterisks (*) indicate a statistically significant difference between curves as determined by the log-rank test ($P < .05$). Figures in parentheses at the bottom indicate number of patients left at risk at that time of follow-up.

facilitating decision making with regard to optimal restoration of LV geometry and size. In the beating ventricle it is easy to assess local wall function by palpation. We have not found an intraventricular balloon to be helpful, although its use has been recommended by centers performing reconstruction in an arrested heart.[4] We agree with Suma and associates[12] that in akinetic aneurysms the limits of scarring are not always well demarcated, nor is the extent of endocardial scar a reliable indicator of the border between functioning and nonfunctioning wall. With alternative repair techniques it may be difficult, in some cases, to determine the exact size and location of patch to be used. Recently, Menicanti and Di Donato[13] described a modification of the Dor procedure, for use in large apical aneurysms, that involves plication of the distal inferior wall before patch placement. Our approach in such cases is to patch the aneurysmal septum and extend the free wall excision over the apex. After removal of the thinned nonfunctioning wall, the length of the residual opening (elliptical in shape) is plicated by placing mattress suture bites wider on the tissue edges than on the felt strips. Completion of the repair locates the new "apex" of the LV next to the RV apex and the conical shape of the LV is restored (Figures 2 and 3). We also tailor the excision and suture line to leave the left anterior descending coronary artery for revascularization. Septal branches may provide collaterals for effective deliv-

TABLE 1. Multivariable predictors of poor 5-year result by means of Cox regression analysis in patients undergoing ventricular reconstruction

Variable	Risk ratio	95% CI	χ^2	*P* value
EF <20%	2.12	1.3–3.5	8.6	.0034
CHF	2.07	1.3–3.4	8.1	.0045
VT	1.94	1.2–3.1	7.4	.0066
Hypertension	1.79	1.0–3.0	4.5	.0336

EF, Ejection fraction; *CHF,* congestive heart failure; *VT,* ventricular tachycardia.

ery of retrograde cardioplegia, and revascularization of even a small portion of the septum may help improve short- and long-term results.[14,15]

We do not perform ventricular reconstruction in patients whose wall motion abnormalities occur in areas of relatively normal wall thickness. We prefer to revascularize these areas and have achieved good results with this approach, even in patients with severely depressed ventricular function (EF < 20%).[16]

This series includes patients with a variable extent of akinetic and dyskinetic scar in both anterior and posterior locations. A high proportion of patients had multivessel coronary disease, advanced LV dysfunction, and ventricular arrhythmias, factors found in other series to be related to increased operating room mortality.[4,5,13,14,17] Nevertheless our hospital mortality was low, 2.8% compared with those in other series, which range from 7% to 13%.[4,6,13,14] Clearly, case selection and operative technique may account for these differences.

After ventricular reconstruction, close follow-up and adjustment of medications is necessary to optimize results. During follow-up, 13 patients underwent additional procedures for increasing heart failure, including 8 transplants and 2 mitral valve replacements. Five-year survival was 82%, which can be compared with survival in other reported series at 2 years, 82% to 86%,[6,18] and at 5 years, 58% to 70%.[5,14,17,19]

We defined poor 5-year result as the need for transplantation or repeated hospitalization for congestive heart failure. Multivariable predictors of poor 5-year outcome were EF less than 20%, congestive heart failure, ventricular tachycardia, and hypertension (Table 1). In patients with volume data, marked ventricular enlargement also predicted poor outcome: a poor result was obtained in 21 of 78 (33%) with a large heart versus 11 of 59 (19%) with a small heart ($P = .080$). Similar relationships between heart failure, decreased EF, increased ventricular size, and poor long-term result have been reported by others.[4,5,13,17]

In this series, objective evidence of improved LV function after the operation was provided by MUGA or MRI data. The average increase in EF of 10% ± 9% ($P < .000$) is similar to that of 10% to 14% reported in other series.[4,6,13,14] Unfortunately, reliable postoperative information regarding volume and shape was only available with the use of MRI in a small subgroup. Postoperatively, the mean EDVI decreased by 30 ± 38 mL/m^2 ($P < .000$) and mean ESVI decreased by 32 ± 39 mL/m^2 ($P < .000$). These values are similar to those reported by Athanasuleas for the RESTORE Group.[4]

In prior reports, operative mortality was increased in patients with akinesis versus dyskinesis, and it has been suggested that patients with akinesis benefit less in terms of symptom relief, improvement in EF, and long-term survival.[20,21] In this series there was no difference between akinetic and dyskinetic groups with respect to patient characteristics including time delay between infarct and repair. Five patients in the akinetic group died in the hospital, with no deaths in the dyskinetic group ($P = .158$). There was no difference in the need for inotropic drugs or intra-aortic balloon pumping. Among survivors, more patients in the akinetic group had improved symptoms: 58 of 69 (84%) versus 35 of 53 (66%) $P = .031$; however, the postoperative change in EF (10% ± 11% versus 10% ± 10% [$P = .953$]) and 5-year survival were similar: 89% versus 78% ($P = .168$). Dor and associates[9] also showed no difference between the 2 groups.

This report includes 32 patients with posterior repair. Compared with the anterior group, these patients were older, had angina less often, smaller areas of abnormal wall motion as defined by percent asynergy, and better preoperative EF (Appendix 1). At surgery, only 1 posterior reconstruction involved a patch septoplasty but in 4 (13%), a small pericardial patch was used adjacent to the mitral annulus to reinforce the linear closure as previously described.[8] This group had more valve interventions than the anterior group: 3 of 37 (9%) versus 3 of 253 (1%) ($P = .020$). Tavakoli and associates[22] reported worse results with posterior repairs. In our series all patients with posterior repair survived and had similar improvement in symptom class and change in EF compared with anterior repairs. Long-term survival was excellent: 97%, 97%, and 90% at 1, 5, and 10 years (a difference that almost reached statistical significance compared with the anterior group $P = .059$).

In considering patients for LV reconstruction the issue of concomitant MR is important. Preoperative Doppler echocardiography often indicates 2+ MR when none was suspected on the angiogram. Early in this series, we were reluctant to add a valve intervention, and in 129 (45%) patients with 2+ MR or more, nothing was done to the valve. In these cases we believed that LV reconstruction would improve mitral valve function, which it did in 74 (57%); MR improved by at least 1 grade.[23] Possible mechanisms for this improvement include (1) decreased annular dilatation caused by decreased ventricular size, (2) im-

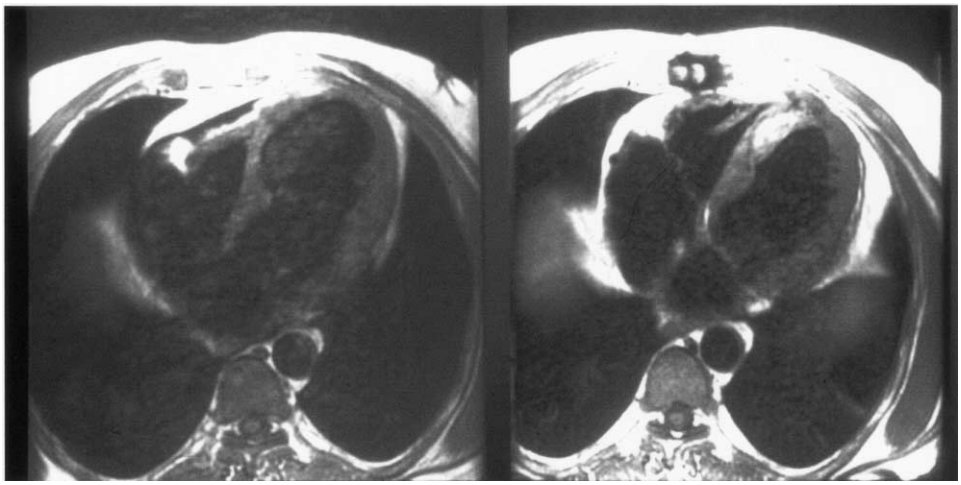

Figure 5. Preoperative *(left)* and postoperative magnetic resonance imaging. Preoperatively, an anteroapical infarct has resulted in thinning of the distal septum. The distal portion of the ventricle is globular in shape. After repair with a septal patch, there is restoration of a more normal conical shape to the ventricle.

proved papillary muscle function related to revascularization, or (3) realignment of papillary muscles related to improved geometry after repair. Recently we have replaced the mitral valve in 3 patients with 4+ MR. For those with 2+ to 3+ MR we have completed the ventricular reconstruction and then reassessed MR with transesophageal echocardiography. For persistent 3+ MR we performed an annuloplasty using an undersized ring as recommended by Bolling and coworkers,[24] or using a patch annuloplasty technique that we described for posterior repairs.[25] Whether these interventions will further improve long-term results needs to be assessed.

In ischemic cardiomyopathy, ventricular arrhythmias are a major source of morbidity and mortality. The value of revascularization in decreasing arrhythmia recurrence needs to be stressed. The CABG Patch Trial showed that use of an implantable cardioverter-defibrillator provided no additional benefit compared with revascularization alone with respect to arrhythmia recurrence and long-term survival in patients with poor EF (<35%) and an abnormal signal averaged electrocardiogram.[26] When coronary artery bypass grafting is combined with ventricular reconstruction, the potential for controlling arrhythmias should be even greater. Our experience using a mapping balloon in patients with recurrent ventricular tachycardia demonstrated that ease of arrhythmia induction was related to mechanical loading conditions.[10] Other investigators have confirmed that myocardial stretch is arrhythmogenic.[27] Therefore, any procedure that restores ventricular volume and size toward normal (such as ventricular reconstruction) should help prevent ventricular arrhythmias.

Mapping studies in patients with anteroapical scar showed that the arrhythmia substrate was located in the border zone between scar and normal endocardium on the ventricular septum.[28] In patients with septal scarring, we performed a visually directed endocardial excision and peripheral cryoablation. Our series includes 108 patients with preoperative ventricular tachycardia, which tended to occur in dilated hearts: 45 of 101 (45%) with a large anterior scar versus 23 of 101 (23%) with a small anterior scar, $P = .002$. Postoperatively, these patients had an electrophysiologic study and those with inducible or spontaneous ventricular tachycardia were discharged on amiodarone or received an implantable cardioverter-defibrillator (n = 9). Even though ventricular tachycardia predicted a poor outcome at 5 years, ventricular arrhythmias during follow-up were only rarely a problem. Excluding patients receiving an implantable cardioverter-defibrillator, freedom from ventricular tachycardia or sudden death was 99%, 97%, and 94% at 1, 5, and 10 years. These results can be compared with those of Wellens and associates,[29] who used a visually directed ablation procedure to achieve at 4 years an 84% freedom from ventricular tachycardia and a 57% survival. The combination of revascularization, LV reconstruction, and visually directed ventricular tachycardia ablation appears to be very effective in preventing arrhythmias.

Recently, Buckberg,[30] has proposed that in patients with ischemic cardiomyopathy, ventricular shape may have an important influence on function and prognosis. In our experience, postoperative MRI has shown return of shape toward normal (Figures 2 and 5), yet the mean diastolic eccentricity index (which is supposed to reflect shape change and which would be expected to increase postoperatively) actually decreased 0.78 ± 0.06 versus 0.74 ± 0.11, $P = .039$. Dor has reported similar results; mean preoperative diastolic eccentricity index 0.70 ± 0.09 decreased

postoperatively to 0.44 ± 0.18. This seems counterintuitive and requires some explanation. Ventricular shape in these patients is asymmetric, and after anteroapical infarction the major shape change occurs in the distal third to half of the ventricle. Standard measures used to assess shape change, the sphericity index or eccentricity index, are based on two end-diastolic dimensions, the long axis and the short axis diameter at the upper third of the ventricle. With progressive adverse remodeling, the magnitude of change in the short axis diameter depends on size, location, and compliance of the infarct and whether or not there is associated MR. For example, 1 patient in this series had an EDVI of 289 mL/m^2 and an ESVI of 259 mL/m^2, yet the short axis diameter was only 54 mm in diastole and 41 mm in systole. We believe that in such patients the shape should be evaluated in a way that draws attention to the distal half of the ventricle, where the major shape changes occur. This would be particularly appropriate in attempting to compare results achieved with different surgical repair techniques.

Limitations of the Study

In this single center study, we used specific selection criteria to choose patients for LV reconstruction including the requirement for relative wall thinning. Whether application of the patch exclusion technique as recommended by the RESTORE Group to exclude akinetic areas of normal thickness will lead to better results than those currently achieved in such patients with revascularization alone, remains to be seen. Another limitation of this series is the lack of postoperative studies in most patients, which are needed for detailed analysis of changes in ventricular volume and shape after repair. Nevertheless, the available MRI data showed significant changes in ESVI, which are comparable with those reported by the RESTORE Group (mean decrease in ESVI 32 ± 39 mL/m^2 versus 34 ± 42 mL/m^2).[4] Further follow-up will be needed to relate the observed changes in volume and shape to long term outcome.

On the basis of our results, we recommend an aggressive approach to revascularization and ventricular reconstruction in patients with coronary disease and poor LV function. We believe that areas of akinesis or dyskinesis associated with relative thinning should be excised to restore ventricular size and shape toward normal and to prevent progressive adverse remodeling. If repair is delayed, contractile function of the residual viable muscle may deteriorate with decrease in EF, increase in wall stress, and chamber dilatation, which may be associated with a less satisfactory surgical result.[13]

Like Dor[9] and Di Donato and Menicanti,[13] we recommend early repair because surgical risk in these patients is low. We suggest that patients with akinesis or dyskinesis should be observed closely and considered for surgical treatment when signs of decompensation first occur, such as increasing symptoms with optimal medical management or in asymptomatic patients if there is evidence of increasing ventricular volume or MR. Surgery should be considered before these patients reach the stage when transplantation is the only reasonable option. As this series demonstrates, in selected patients ventricular reconstruction by a modified linear closure technique plus patch septoplasty, when indicated, can be done in the beating heart with low operative mortality. The procedure provides good symptomatic improvement and excellent 5-year survival. The role of concomitant valve procedures in those with MR requires further investigation.

References

1. Jatene AD. Left ventricular aneurysmectomy. *J Thorac Cardiovasc Surg*. 1985;89:321-31.
2. Cooley DA. Ventricular endoaneurysmorrhaphy: a simplified repair for extensive postinfarction aneurysm. *J Card Surg*. 1989;4:200-5.
3. Dor V, Saab M, Coste P, Kornaszewski M, Montiglio F. Left ventricular aneurysm: a new surgical approach. *J Thorac Cardiovasc Sug*. 1989;37:11-9.
4. Athanasuleas CL, Stanley AW Jr, Buckberg GD, Dor V, Di Donato M, Silver W, RESTORE Group. Surgical anterior ventricular endocardial restoration (SAVER) for dilated ischemic cardiomyopathy. *Semin Thorac Cardiovasc Surg*. 2001;13:448-58.
5. Di Donato M, Toso A, Maioli M, Sabatier M, Stanley AW Jr, Dor V, RESTORE Group. Intermediate survival and predictors of death after surgical ventricular restoration. *Semin Thorac Cardiovasc Surg*. 2001; 13:468-75.
6. Suma H, Isomura T, Horii T, Hisatomi K. Left ventriculoplasty for ischemic cardiomyopathy. *Eur J Cardiothorac Surg*. 2001;20:319-23.
7. Mickleborough LL, Maruyama H, Liu P, Mohammed S. Results of left ventricular aneurysmectomy with a tailored scar excision and primary closure technique. *J Thorac Cardiovasc Surg*. 1994;107:690-8.
8. Mickleborough LL. Left ventricular aneurysm: modified linear closure technique. In: Cox JL, Sundt TM III, editors. Operative techniques in cardiac and thoracic surgery: a comparative atlas. Vol II. Philadelphia: WB Saunders; 1997. p. 118-31.
9. Dor V, Sabatier M, Di Donato M, Montiglio F, Toso A, Maioli M. Efficacy of endoventricular patch plasty in large postinfarction akinetic scar and severe left ventricular dysfunction: comparison with a series of large dyskinetic scars. *J Thorac Cardiovasc Surg*. 1998;116:50-9.
10. Di Donato M, Sabatier M, Dor V, et al. Effects of the Dor procedure on left ventricular dimension and shape and geometric correlates of mitral regurgtitation one year after surgery. *J Thorac Cardiovasc Surg*. 2001;121:91-6.
11. Mickleborough LL, Usui A, Downar E, Harris L, Parson I, Gray G. Transtrial balloon technique for activation mapping during operations for recurrent ventricular tachycardia. *J Thorac Cariovasc Surg*. 1990; 99:227-33.
12. Suma H, Isomura T, Horri T, et al. Nontransplant cardiac surgery for end-stage cardiomyopathy. *J Thorac Cardiovasc Surg*. 2000;119:1233-45.
13. Menicanti L, Di Donato M. The Dor procedure: What has changed after fifteen years of clinical practice? *J Thorac Cardiovasc Surg*. 2002;124:886-90.
14. Pasini S, Gagliardotto P, Punta G, et al. Early and late results after surgical therapy of postinfarction left ventricular aneurysm. *J Cardiovasc Surg*. 1998;39:209-15.
15. Sinatra R, Macrina F, Braccio M, et al. Left ventricular aneurysmectomy; comparison between two techniques: early and late results. *Eur J Cardiothorac Surg*. 1997;12:291-7.
16. Mickleborough LL, Carson S, Tamariz M, Ivanov J. Results of revascularization in patients with severe left ventricular dysfunction. *J Thorac Cardiovasc Surg*. 2000;119:550-7.
17. Cosgrove DM, Lytle BW, Taylor PC, et al. Ventricular aneurysm resection: trend in surgical risk. *Circulation*. 1989;79(Suppl 1):97-101.

18. Grossi EA, Chinitz LA, Galloway AC, et al. Endoventricular remodeling of left ventricular aneurysm: functional, clinical, and electrophysiological results. *Circulation*. 1995;92(Suppl 2):98-100.
19. Shapira OM, Davidoff R, Hilkert RJ, et al. Repair of left ventricular aneurysm: long-term results of linear repair versus endoaneurysmorrhaphy. *Ann Thorac Surg*. 1997;63:701-5.
20. Couper GS, Bunton RW, Birjiniuk V, et al. Relative risks of left ventricular aneurysmectomy in patients with akinetic scars versus true dyskinetic aneurysms. *Circulation*. 1990;82(Suppl 4):248-56.
21. Yannikas J, MacIntyre W, Underwood D, et al. Prediction of improvement in left ventricular function after ventricular aneurysmectomy using Fourier phase and amplitude analysis of radionuclide cardiac blood pool scans. *Am J Cardiol*. 1985;55:1308-12.
22. Tavakoli R, Bettex D, Weber A, et al. Repair of postinfarction dyskinetic LV aneurysm with either linear or patch technique. *Eur J Cardiothorac Surg*. 2002;22:129-34.
23. Mickleborough LL, Carson S, Ivanov J. Repair of dyskinetic or akinetic left ventricular aneurysm: Results obtained with a modified linear closure. *J Thorac Cardiovasc Surg*. 2001;121:675-82.
24. Bolling SF, Pagani FD, Deals GM, Bash DS. Intermediate term outcome of mitral reconstruction in cardiomyopathy. *J Thorac Cardiovasc Surg*. 1998;115:381-6.
25. Konstantinov I, Mickleborough LL, Graba J, Merchant N. Intraventricular mitral annuloplasty technique for use with repair of posterior left ventricular aneurysm. *J Thorac Cardiovasc Surg*. 2001;122: 1244-7.
26. Bigger JT, Coronary Artery Bypass Graft (CABG) Patch Trial Investigators. prophylactic use of implanted cardiac defibrillators in patients at high risk for ventricular arrhythmias after coronary artery bypass graft surgery. *N Engl J Med*. 1997;337:1569-75.
27. Koilpillai C, Quinones MA, Greenberg B, et al. Relation of ventricular size and function to heart failure status and ventricular dysrhythmia in patients with severe left ventricular dysfunction. *Am J Cardiol*. 1996; 77:606-11.
28. Downar E, Kimber S, Harris L, et al. Endocardial mapping of ventricular tachycardia in the intact human heart. II. Evidence for multiuse reentry in a functional sheet of surviving myocardium. *J Am Coll Cardiol*. 1992;20:869-78.
29. Wellens F, Geelen P, Demirsoy E, et al. Surgical treatment of tachyarrhythmias due to postinfarction left ventricular aneurysm with endoaneurysmorrhaphy and cryoablation. *Eur J Cardiothorac Surg*. 2002;22:771-6.
30. Buckberg GD. The helix and the heart: basic science review. *J Thorac Cardiovasc Surg*. 2002;124:863-83.

Discussion

Dr Lorenzo A. Menicanti *(Segrate, Italy).* This paper is an excellent review of Dr Mickleborough's personal experience. It demonstrates the great benefits of volumetric reduction surgery and confirms the results obtained by other surgical groups. We completely agree with the general surgical philosophy presented in this paper. There are several important questions that I would like to discuss with the author, as well as the idea that these procedures should be standardized as much as possible.

In your manuscript, you consider patients with severe pulmonary hypertension, RV dysfunction, and diffuse disease of the coronary arteries to be ineligible. These patients are in the worst condition, with increased operative mortality, but the procedure may be the only option they have, and probably we must give them a chance. Can you define more precisely the dimensions of these parameters?

The MR in your paper is a relative contraindication. You present MR of more than 2+ in 13% of the cases, but only 2% are treated. Can you define your present strategy when 3+ MR is present or when MR is moderate but with an enlarged annulus?

Dealing with the surgical technique, in the RESTORE Group experience, the results with the beating and the arrested heart were compared but no differences were found. Do you think that this is really important in the procedure? In my understanding, the beating heart technique can be harmful when a recent infarction is treated and the tissues are fragile or when the mitral valve is approached.

The septoplasty in your presentation was performed in 22% of the cases. How do you judge the septal involvement before going into the operating room?

Some authors demonstrate that excessive reduction of the longitudinal diameter can result in diastolic dysfunction of the LV. Therefore, preserving the normal relationship between longitudinal and transverse diameter can be of importance. How do you treat the ventricle that has a transitional zone at the base of the papillary muscle involving the inferior wall to avoid a spherical cavity?

In your paper you said that excision is planned to remove as much nonfunctioning wall as possible. The risk is that too small a cavity will be created. Do you have some guideline to avoid this complication?

Your experience covered a period of 20 years. Did you notice any differences in patients' characteristics in the most recent period when primary angioplasty and thrombolysis are so frequent?

Finally, did the introduction of MRI change in some way your strategy or your results?

Dr Mickleborough. Thank you, Dr Menicanti, for those kind remarks. You and Dr Dor have a similar experience using slightly different techniques, but I believe that our philosophy overlaps greatly. I will try to answer all of your questions.

As far as patient selection is concerned, if a patient's pulmonary artery pressures are systemic or near systemic, I am not as brave as you are: I cannot get such patients through this operation. I would consider patients with severe RV failure with recurrent hepatic congestion or coagulopathy to be inoperable with this approach.

Diffuse coronary artery disease is a very relative thing. Many patients in this series had nonvisualized vessels, but about 90% of the time, even when a vessel is completely nonvisualized, when you explore the area you will find something you can graft. When I refer to inoperable coronary disease, I am talking about patients whose vessels you can see, but they are moth-eaten all the way down. There is no good place to do a graft, and all three distributions are affected. I consider such patients with nongraftable coronary artery disease and very poor ventricular function too much of a challenge and inoperable.

With respect to MR, when I started doing this in the 1980s, I was very reluctant to do a mitral valve procedure in addition to map-guided ablation of ventricular tachycardia, LV reconstruction, and bypass surgery. In the past 10 years I have become much more willing to take on patients with 4+ MR or 3+ MR, and at the present time my recommendation would be that anyone who has 3+ or 4+ MR needs a valve procedure. In my hands, if they have 4+ MR they get a valve replacement, and if they have 3+ MR, I will do a valvuloplasty. If they have 2+ MR going into the procedure, I will do my reconstruction and then do a transesophageal intraoperative assessment. In the past, we have not had a patient in whom the MR has gotten worse, but if that should

happen and the MR was then 3+, I would go back and do a valvuloplasty. I do not think these patients should be left with 3+ MR.

Do I think the beating heart is important? That ties in with another of your questions about how to maintain ventricular volume after the repair. Nature is a wonderful thing. When we are referred patients with a prior myocardial infarction, nature has already selected who will survive and who will not. If a patient makes it to our operating room, God has been generous to him or her and has left enough functioning ventricle behind so that if I do not make a mistake and get too greedy with my resection, the patient will not end up with too small a cavity. In my experience, the location of the papillary muscles is a very useful guide. In doing the resection, stay away from the base of the papillary muscles. Leave a little bit of thin wall in, if you have to. Do not involve the papillary muscles in the resection, because if you start relocating the papillary muscles in these types of patients, you may very well end up with too small a cavity.

In determining the limits of the resection, as in valve surgery, experience counts and the decision should be based on your gestalt of the situation. To make these decisions, there is no "correct" formula. Your group uses a balloon, which may be an excellent device. I do not find it helpful, because in the beating heart, the balloon will not stay where I want it anyway. So I use my experience and visual assessment to ensure an adequate size in the cavity that will result from the repair.

With respect to the septum, before I had MRI, I had to rely on visual assessment at the time of the operation. When you open up such a heart and look at the septum, you can tell whether it is just scarred or whether it is scarred and bowed into the RV cavity. In the latter case, you need to do a septoplasty. MRI is a wonderful technique, and with it you can tell ahead of time exactly which patient has a thinned aneurysmal septum and needs to have a septoplasty and which patient does not.

As far as the short and long axis ratio is concerned, the sphericity index is a very good index to assess symmetrical change in ventricular shape, but it is terrible, in my opinion, for assessing the changes that occur in these patients. With anteroapical infarcts, most of the shape changes occur at the apex, not where the papillary muscles insert and not where we measure short axis. I think the sphericity index is the wrong index to be looking at when judging shape change. Do I have a better one? Not yet, but I am working on it.

This is only a 20-year experience, and we see fewer of the huge dyskinetic anteroapical aneurysms than we used to. I think that is because of better earlier intervention by our cardiology colleagues.

Dr Aubrey C. Galloway *(New York, NY).* Dr Mickleborough, this is an excellent series and a group of patients whom everyone will be seeing more frequently as our surgical population continues to age and we operate on more patients with severely depressed LV function.

As a follow-up on the earlier question related to concomitant mitral valve repair, it appears that you recommended mitral repair or replacement in patients with 3+ or 4+ MR, which is what we would also advocate. Do you have any data that support this recommendation? The recommendation certainly seems intuitively obvious, based on other studies evaluating the impact of MR on patients with poor ventricular function, showing that patients with 3+ or 4+ MR have better survival and fewer symptoms if the MR is corrected. In your series, did you see a progressive deterioration in the patients with moderate MR in whom valve repair was not performed?

Dr Mickleborough. That is a very good question. During follow-up, there were 69 late deaths, and 39 of those deaths were due to progressive congestive heart failure. Most of those patients had progressive MR as part of the heart failure. Yes, I think this is a very important issue.

We previously reported results achieved with revascularization alone in patients with moderate MR. In those patients, the untreated valve did not seem to matter so long as ventricular function was good. In other words, the patients who had 1+ or 2+ MR and had a grade 1 or 2 ventricle lived just as long as the ones who had no MR. However, the subset that had grade 3 or 4 ventricles and 2+ MR did not live nearly as long.

The ventricle will always be tied in with the mitral valve apparatus and vice versa. I offer the following guidelines: In grade 3+ or 4+ MR, something must be done, whether it is replacement or repair. Grade 2+ MR should be reassessed after the reconstruction has been completed. If the regurgitation is the same or less, the valve should be left alone and I think the 5-year survival will be good. If it is worse, the valve should be fixed.

Appendix 1. Patient subgroups

	Anterior (n = 253)	P	Posterior (n = 32)	Akinetic (n = 97)	P	Dyskinetic (n = 64)
Preoperative clinical variables						
Age (y)	60 ± 10	.001*	66 ± 7	61 ± 9	.116	59 ± 10
Male sex	212 (84%)	.452	25 (78%)	79 (81%)	.522	55 (86%)
Angina	145 (57%)	.039*	12 (35%)	54 (56%)	.418	40 (62%)
CHF	156 (62%)	.569	18 (56%)	64 (66%)	.408	38 (59%)
VT	91 (63%)	.081	17 (53%)	32 (33%)	1.000	21 (33%)
Diabetes	38 (19%)	1.000	5 (17%)	76 (78%)	.402	54 (84%)
Hypertension	95 (47%)	.387	18 (57%)	50 (52%)	.617	30 (47%)
PVD	20 (10%)	.712	4 (13%)	13 (13%)	.598	6 (9%)
Symptom class						
I	8 (3%)	.780	2 (6%)	2 (2%)	.207	5 (8%)
II	34 (13%)		5 (16%)	14 (14%)		9 (14%)
III	85 (33%)		11 (34%)	38 (39%)		18 (28%)
IV	126 (50%)		14 (44%)	43 (44%)		32 (50%)
Interval from infarct (mo)	55 ± 72	.408	43 ± 59	58 ± 75	.903	56 ± 73
Catheterization data						
Coronary disease						
Single	40 (16%)	.903	4 (13%)	14 (14%)	.634	12 (19%)
Double	78 (31%)		11 (34%)	34 (35%)		19 (30%)
Triple	133 (53%)		17 (53%)	49 (51%)		33 (52%)
Ejection fraction						
40%	13 (5%)	.022*	4 (13%)	4 (4%)	.257	6 (9%)
20%-40%	97 (38%)		19 (59%)	41 (42%)		22 (34%)
20%	143 (57%)		9 (29%)	52 (54%)		36 (56%)
Mean EF	27 ± 10	.006*	30 ± 12	28 ± 9	.579	260 ± 10
MR 2+ or more	126 (44%)	.113	18 (56%)	56 (58%)	.488	39 (61%)
EDVI (mL/m^2)	166 ± 53	.913	165 ± 50	166 ± 57	.968	166 ± 47
ESVI (mL/m^2)	104 ± 51	.993	104 ± 38	102 ± 52	.598	107 ± 50
Asynergy (%)	54 ± 17	.048*	46 ± 21	54 ± 18	.910	54 ± 15
Intraoperative variables						
Crossclamp time	62 ± 23	.008*	74 ± 33	60 ± 24	.400	64 ± 25
Pump time	171 ± 51	.845	173 ± 53	160 ± 41	.200	169 ± 47
CABG (%)	233 (92%)		29 (91%)	94 (97%)		59 (92%)
Average no. grafts	2.8 ± 1.3	.688	2.7 ± 1.2	3.0 ± 1.2	.570	2.9 ± 1.5
LITA use	127 (50%)	.575	14 (44%)	62 (64%)	.410	36 (56%)
VT ablation	110 (44%)	.056	8 (25%)	36 (37%)	.154	31 (48%)
Patch septoplasty	63 (25%)	.003*	1 (3%)	30 (31%)	.399	24 (38%)
Valve procedure	3 (1%)	.020*	3 (9%)	2 (2%)	.518	0
Results						
Inotropic agents	128 (56%)	.178	13 (41%)	51 (53%)	.620	30 (47%)
IABP	46 (23%)	.103	3 (9%)	18 (19%)	.834	12 (19%)
Mortality	8 (3.2%)	.604	0	5 (5%)	.158	0
Improved symptoms	122/161 (76%)	.472	18/26 (69%)	58/69 (84%)	.031*	35/53 (66%)
Change in EF (%)	10 ± 9	.186	6 ± 16	10 ± 10	.816	10 ± 10
Good 5-y result	133/181 (74%)	.031*	21/22 (96%)	48/66 (73%)	.278	32/39 (83%)

CHF, Congestive heart failure; *VT*, ventricular tachycardia; *PVD*, peripheral vascular disease; *EF*, ejection fraction; *EDVI*, end-diastolic volume index; *ESVI*, end-systolic volume index; *CABG*, coronary artery bypass grafting; *LITA*, left internal thoracic artery; *IABP*, intra-aortic balloon pump.